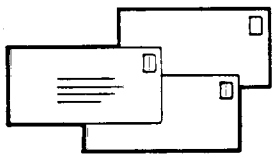

Letters to the Editor

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Letters should be submitted in duplicate, double-spaced (including references), and should not exceed 400 words.

Risk to Passive Smokers

Sandler[1] is a follow-up to an earlier publication by Helsing.[2] It raises issues that are in need of a response. These papers are a result of a 1963 survey in Maryland where health-related questions were asked, including smoking status. In 1975, death records were reviewed to determine if "passive smokers" were at more risk than nonsmokers who were not exposed to environmental tobacco smoke (ETS) in the home.

The introduction indicates the authors were conducting an "evaluation" of the population in question. However, there is no clear statement of the purpose of the investigation or definition of a problem.

The 1988 Helsing paper[2] reported a higher relative risk (1.31 for men and 1.24 for women) for deaths due to arteriosclerotic heart disease nonsmokers married to smokers versus nonsmokers married to nonsmokers. The Sandler study[1] reports a higher relative risk of death from all causes in ETS-exposed persons: 1.17 for men and 1.15 for women. The authors[1] admit the results could be influenced by confounding factors such as different lifestyle, including alcohol consumption in those in the group exposed to ETS. "Finally, with relative risks as low as those observed here, and with such weak dose-response, one must consider that other biases are responsible for these findings."

Significantly, Sandler fails to acknowledge the serious deficiencies and limitations of death certificates, his principal source of data. This could easily confound the intent of the study especially in those cases where death was the result of a terminal malignancy which was not confirmed by autopsy/histology.

Examination of Table 4 indicates another problem that could be termed "selective weighting" of population components. The paper ultimately draws attention to respiratory diseases data in this table. Examination of these numbers shows them to be very limited. The small sample size precludes effective and meaningful statistical evaluation.

Sandler[1] fails to recognize the 1988 report on lifestyle factors by Koo[3] that demonstrates confounding factors present in such evaluations of ETS. The reviews of epidemiological flaws as pointed out by Uberla with regard to ETS studies and by Feinstein[5] with regard to faulty methodology used in epidemiological studies in general are also ignored.

Overall, the Sandler[1] paper should have been rejected for publication on the basis that it is poor science and inadequately designed and undertaken to address the issue presented in the title. It adds little if anything to the body of data needed to allow a justifiable determination of the relationship between exposure to ETS and risk of mortality.

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Response from Sandler and Comstock

In his letter, Holcomb has alleged serious deficiencies in our paper[1] on mortality in nonsmokers who lived with smokers. He wrote that because we "evaluated" mortality in nonsmokers living with smokers, we had no clear purpose in conducting our study. To the contrary, the study was designed specifically to obtain evidence bearing on possible health effects associated with living with smokers. We did not collect independent data for this purpose, but selected data from several existing sources that would best address this question. We do not claim to have a definitive test of our hypothesis and feel that our results have been appropriately qualified.

With respect to Holcomb's denigration of death certificate information, few will join him in challenging their usefulness in ascertaining total mortality in this or other developed countries. Death certificates do have limitations with respect to causes of death, and especially other conditions present at death. These limitations have rarely, however, misled the scientific world in searches for associations.

We reported that the overall death rate for nonsmokers who lived with smokers was increased relative to that for nonsmokers who were not exposed.[1] Certainly, Holcomb would agree that a death certificate indicates that a death has occurred. Helsing, *et al.*[2] clearly demonstrated an association between deaths attributed to arteriosclerotic heart disease and exposure to household tobacco smoke. Others will have to judge whether or not the association is causal in nature. Current evidence suggests causality as more likely than confounding. While Holcomb's attention was drawn to findings regarding respiratory diseases, this was not so singled out by us. Nonetheless, such an effect is consistent with what is known about smoking and lung disease.[3]

As we stated in our paper, we took advantage of data that had been collected for other purposes. For this rea-

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son we were not able to rule out alternative explanations for our findings to the extent we would have liked. We found few differences, however, in our study[4] or in other reports[5,6] that would account for our findings. Koo, *et al*,[7] compared 70 nonsmoking women with nonsmoking husbands to 66 nonsmoking women whose husbands smoked. They were able to shed very little additional light on the question of differences between smoking and nonsmoking households. In fact, many of the differences they observed, such as increased respiratory illnesses, use of medications and medical procedures, and adverse pregnancy outcomes, were likely to have been the result of living with smokers rather than an indication of fundamental differences between the groups.

We believe the findings of our study were worthy of publication. The study raises the possibility that living with smokers may have lasting detrimental effects on health. We hope our findings will encourage additional studies and serious dialogue about this issue.

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Environmental Tobacco Smoke and Legal Issues

The Byrd, *et al*, review article[1] attempts to form a bridge between medical data pertaining to environmental tobacco smoke (ETS) and legal issues evolving from regulation of ETS. This bridge is needed to improve our understanding and management of ETS in ambient air.

To provide a scientific basis for their position, these authors must relate ETS exposure to a medical condition. They must define the causative agent. They must identify the route of exposure including concentration. There must be an exposure duration. If these are lacking, the resulting conclusions are compromised. This article considers the chemical composition of ETS merely by a discussion of chemical differences between mainstream and sidestream smoke.

When actual human ETS exposures are considered, this review article does not say much about typical concentrations of chemical constituents to which nonsmokers are exposed. Therefore, we never know the human dosage of any chemical constituent in ETS. There is no consideration of duration of exposure. All ETS studies performed to this date suffer from the absence of a reliable measurement of ETS exposure. Unfortunately, these authors ignore this research problem as well.

The Byrd, *et al*, review suggests that ETS causes respiratory distress in children, lung cancer, and cardiovascular/respiratory health problems. The children's studies are presented without mentioning the ambiguity of many results, the presence of confounding factors, and the subject stress introduced by the study procedures.

The Surgeon General's 1986 report[2] calls ETS a "plausible" cause of lung cancer which is accepted by this review. However, this review does not adequately communicate the conclusions in the body of the Surgeon General's 1986 report wherein much of the supporting research was found to be technically flawed and/or statistically insignificant. These authors also neglect to consider the psychological factors involved in the onset of cardiovascular/respiratory distress.

The literature review in this article is limited and slanted toward the political/personal opinions of the Surgeon General. The evaluation of medical data is brief and does not present the current status of science in this field. Consequently, this effort to link ETS medical data to evolution of legal thought is less than it should be. The remainder of the review is a historical treatment of legal/policy decisions which have addressed ETS.

ETS investigators will benefit from the overview of experienced scientists who apply empirical skills to this bridge area when they write review articles that are comprehensive and objective. The literature is presently overloaded with subjective manipulations of questionable data by review article authors, public health officials, and editors.

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Response from Dr. Byrd

Dr. Halfen raises a number of important issues. The scientific study of passive smoking is barely a decade old. Much future research is necessary to resolve areas of uncertainty. Reliable information needs to be gathered on the fate of environmental tobacco smoke (ETS) in ordinary indoor environments; improved surveys quantifying passive smoke exposure should be developed; and biological studies must be refined to quantify the exposure-dose relationship in nonsmokers.

We disagree, however, with Dr. Halfen's assertion that a specific etiologic chemical needs to be identified in order to establish ETS as a cause of disease. Active cigarette smoking was known to cause lung cancer years before the chemical constituents of tobacco smoke were delineated. Now passive smoking has been shown to cause lung cancer. The goals of future research are to further define the magnitude of the risk and to seek the specific causative agents.

The investigation of passive smoking has become increasingly sophisticated. Recent studies have utilized more rigorous designs and monitoring

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